Editorial and Special Articles

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MINUTES OF EXECUTIVE MEETING

Minutes of a meeting of the Winnipeg members of the Executive of the Manitoba Medical Association, held in the Medical Arts Club Rooms on Wednesday, February 12th, 1936, at 12.30 noon.

Present.

Dr. F. G. McGuinness	Dr. W. E. R. Coad
Dr. D. C. Aikenhead	Dr. W. G. Campbell
Dr. F. W. Jackson	Dr. E. S. Moorhead
Dr. C. W. Burns	Dr. C. W. MacCharles
Dr. W. E. Campbell	Dr. G. S. Fahrni
Dr. A. S. Kobrinsky	Dr. W. W. Musgrove
Dr. F. A. Benner	Dr. C. R. Rice.

(Drs. Fahrni, Musgrove and Rice are representing the M.M.A. on the Committee of Twelve).

Re. Annual Meeting.

Dr. C. W. Burns, convener of the Programme Committee, was asked for a report. Dr. Burns advised as yet he had nothing to report, although he had been in communication with the Dean and Professor of Medicine and Surgery, and would like to know definitely the date of the meeting. As the date of convocation is May 13th, it was moved by Dr. F. A. Benner, seconded by Dr. W. E. R. Coad: That the Annual Meeting be held on the 14th, 15th and 16th of May. —Carried.

Discussion took place as to whether or not any oral examinations in the third and fourth year of medicine might not take place on those dates.

It was moved by Dr. A. S. Kobrinsky, seconded by Dr. C. W. Burns: That the Dean be approached to see if it could not be arranged to have these dates left free. —Carried.

Re. Exhibits at Annual Meeting.

The question of exhibits at the Annual Meeting came up for discussion.

It was moved by Dr. E. S. Moorhead, seconded by Dr. A. S. Kobrinsky: That the President and Secretary be a Committee to make arrangements with Mr. J. G. Whitley in reference to arranging for exhibits.

It was moved by Dr. F. A. Benner, seconded by Dr. W. E. R. Coad: That the President be authorised to appoint a Committee in charge of social functions. —Carried.

Report of Committee on Twelve
Re. Chiropractors' Bill.

Dr. G. S. Fahrni, as a member of the Association on the Committee of Twelve, made a report in reference to the work of the Committee of Twelve. This had to do, of course, with the decision of the Committee to actively oppose an endorsement of the chiropractors' bill at the coming session of legislation. Dr. Fahrni pointed out that two pamphlets had been issued and that all medical men in the Province had been circularized, and asked for support in interviewing members of the legislation. He also reported that a delegation had waited on the Premier, Minister of Health and Public Welfare and Minister of Education, and had been very favorably received. Dr. Fahrni reported that medical students had been interviewed and they had decided to present a petition to the Governments protesting against the passing of the bill.

General discussion followed, and it was moved by Dr. F. A. Benner, seconded by Dr. W. E. R. Coad: That a resolution be sent by the Manitoba Medical Association to the Minister of Health, asking that he actively oppose the passage of this bill, pointing out that the medical profession had co-operated with the Department of Health at all times, and wished to go on record that the Minister should definitely oppose any legislation granting chiropractors a license to take care of the sick, unless they were prepared to come up to the same standards as prescribed by the University in the medical course.

It was moved by Dr. D. C. Aikenhead, seconded by Dr. W. G. Campbell: That this Executive approve and endorse all work already carried out by the Committee, and wished to especially thank Dr. Fahrni for his untiring efforts on behalf of the profession of Manitoba. —Carried.

The meeting then adjourned.

VICTORIAN ORDER OF NURSES

The Annual Meeting of the Victorian Order of Nurses was held in the Medical Arts Club Rooms on February 4th, 1936. This was the 34th annual meeting of the Winnipeg Branch of the Order. It was reported that the nurses had made during the year 12,755 visits and nursed 1,284 cases. The total number of obstetrical cases was 83 and the confinements attended 67. The decrease in the number of confinements attended is apparently due to the increasing proportion of maternity cases which go to hospital. The service of the Victorian Order of Nurses was made use of by 213 physicians in Winnipeg. Demonstrations were given to the graduating classes of the Winnipeg General, Misericordia, St. Boniface and Grace Hospitals.

UNEMPLOYMENT RELIEF REPORTS

The attention of practitioners on the panel in Winnipeg is drawn to the following letter:

February 10th, 1936.

Dr. E. S. Moorhead,
Chairman, Committee on Sociology,
Manitoba Medical Association,
Winnipeg.

Dear Sir:

Might I draw your attention to the fact that some doctors still continue to send reports to this office having insufficient postage.

This has become so serious that the Postal Department has informed the Relief Department that the Relief Department will be held responsible for all postage due on letters or reports which they accept. The only recourse this department has in that case would be to return letters lacking sufficient postage. This the Relief Department does not wish to do as it would cause delay and trouble to doctors.

The Superintendent asked me to take this matter up with you and ask that the doctors be notified that the Relief Department will be forced to return mail with insufficient postage, if this practice is not stopped.

Yours very truly,
UNEMPLOYMENT RELIEF DEPARTMENT
(Signed) H. HARVEY,
Medical Officer.

Department of Health and Public Welfare

NEWS ITEMS

NOTICE

Scarlet Fever Antitoxin.

Connaught Laboratories are now supplying scarlet fever antitoxin standardized in terms of the unit which was adopted by the United States Public Health Service. This unit is equivalent to fifty original neutralizing units as established by the Scarlet Fever Committee, Incorporated. In other words, each U.S.P.H.S. unit of scarlet fever antitoxin neutralizes fifty standard skin-test doses of scarlet fever toxin. The prophylactic dose of this product now stands increased to contain one-third as much antitoxin as is contained in a treatment-dose package, this ratio having been originally two-fifteenths.

Treatment dose	6000 units
Prophylactic dose	2000 units

EAR CONDITIONS FROM THE PREVENTIVE STANDPOINT

(Edmund Price Fowler, M.D., New York City)

The following is the first portion of a report by Dr. Edmund Price Fowler on "Ear Conditions from the Preventive Standpoint." Dr. Fowler's report will be completed in the next issue of "The Review."

Prevention in the medical sense means "to take precautionary measures against," "to forestall or anticipate" injury, deformity, or disease; also, "to stop, ward off, or thwart" injury, deformity, or disease and the anatomical and functional defects following such. All medical and surgical measures are in their essence preventive, whether they be applied before, during, or after the threat or the appearance of the conditions against which they are directed.

Man differs markedly from other animals in his ability to anticipate attack, whether it be from disease or from his fellow man or from his animal, vegetable, or mineral surroundings. He seldom does this on a large scale because there are too many other things to be done to be constantly anticipating some affliction which, after all, may cause but a small percentage of deaths, although considerable individual crippling of function. And so, except in rare instances, nothing will be done in the line of prevention until there are some signs (usually marked signs) of impending or actual disease.

It is well, generally speaking, that man is not constantly anticipating trouble. Over-precaution engenders psychological and even bodily reactions often more undesirable than the trouble itself, and may in fact degenerate into a neurotic fad. The best justification for preventive treatment as applied to ear conditions is exposure to infection or some sign or history of ear abnormality in self or family.

Prevention may be discussed from many angles: i.e., heredity, sex, age, environment, climate, social conditions, health, habits, idiosyncrasy for or against poisons, irritations, and infections, and immunity and reaction thereto. The digestive tract, blood, bones, glands, endocrines, vasomotor mechanisms, and nervous systems all give problems in reference to prevention. The whole subject will be treated under three main headings, detection, diagnosis and treatment.

DETECTION

The most important preventive measure is early detection in infancy, youth, adolescence, middle and even in old age. Early detection must be concerned not only with the patient, but with the relatives, ancestors, and all offspring because, generally speaking, ear disease, like many other diseases, tends to run in families. This does not mean that it is necessarily inherited as such. It does mean that externally and internally children resemble their parents and, as did their forebears, inherit through anatomical and phsiological similarities, similar reaction to, acquisition of, and resistance to disease in similar environments. The inheritance factor is a matter of dominant genes; it is a matter of similar anatomical and physiological set-ups, plus contacts with similar infections, poisons, or traumas, in children and parents. Family histories indicate in general the direction, force, and duration of the hereditary winds, and what they are likely to pick up on their way. I have unearthed as many as nine in one family (seven children and two parents), all of whom showed tendencies to similar ear disease and deafness. It is my personal opinion that deafness in any member of a family should lead us to examine every member of that family, so that in case of positive findings preventive measures may be instituted.

The only advocated preventive measure for inherited tendencies has been the abolition of offspring. A tendency to cataract, tuberculosis, otosclerosis, bad teeth and even flat feet may be inherited. Should we condemn parents with any of these conditions to a childless marriage? After middle life the hearing of a majority of human beings begins to decrease and at an increasing rate with advancing age. Should we therefore advise this majority (or their children) to refrain from bearing offspring? If not, at what age is deafness to be considered a reason for eugenic control? And why?

I believe it is far better to strive for early detection in parent and child and for an early arrest or cure of the condition with proper preventive follow-up, than to expend energy upon unobtainable and questionable eugenic control. Remember that the struggle for existence is the greatest factor in developing the greatest human (or animal) qualities and that overcoming a defect develops a power to succeed to an extreme degree. A defect in the father may therefore bring about an urge to succeed which may be passed on to the son, not necessarily by inheritance, but surely by example. Like father, like son. No people have contributed so much to the cause of the deafened as the deafened themselves. Does this hold true in any other type of lowered function?

DETECTION OF SYMPTOMS

The usual means for detecting ear trouble is the recognition of abnormal signs or symptoms referable to the ear. If such symptoms are acknowledged upon their first appearance and are acted upon intelligently, early preventive measures may obtain. Such symptoms are:

1. Deafness, dullness, heaviness or blockage sensations persisting for more than a few minutes. especially after exposure to cold, swimming, head colds, fatigue, constipation, shock, or other causes of lowered resistance.
2. Asking for the repetition of words or phrases; hearing better in a noisy place (paracusis); hearing two sounds where only one is sounded (diplacusis); inability to locate the direction from which sounds emanate.
3. Tenderness, itching, heat or pain in and about the ear.
4. Tinnitus (autogenous noises in the ear).
5. Vertigo, a sense of unsteadiness or faulty equilibration, nausea, vomiting, jumping of the eyes (nystagmus).
6. Moisture, running or discharge in the external ear canal.
7. Deformity or swelling in or about the ear (red and painful or otherwise).

8. Headache, fever, sweats, chills, facial paralysis; and many allergic or neuological symptoms correlated to any of the above.

Of course any of these symptoms may be due to other than ear disease and many of them not to any disease. As their detection comes later and later, it is less and less useful from the preventive standpoint.

HEARING TESTS

Early detection is dependent upon early elicitation and recognition of abnormal signs and symptoms. School examinations are now far ahead of anything in the past due to the audiometric tests instituted and largely carried out in New York under the supervision of the New York League for the Hard of Hearing, and recently also under the auspices of the Board of Education. Over 600,000 children have been examined from March, 1934 to April, 1935. Each class, up to 40 pupils, is first tested by the 4A phonograph audiometer. Those showing a loss of 9 db* or more are considered subnormal and after a retest of the borderline cases are tested by the 2A audiometer, by air conduction and by bone conduction. This is the first time that such a complete examination has been accomplished on a large scale.

Preliminary to the hearing test a careful history is obtained, and after the test a comprehensive otological examination. A follow-up system is put into operation which endeavors to continue observation or treatment. A great defect is the inability to obtain the continued co-operation of parents, physicians and clinics, even under repeated provocation and with a definite history of family ear disease or deafness. Another defect is that children with low normal hearing are not given an otological examination. Many such have ear disease and go on to severe deafness. The only way to obviate the poor results now obtained is to test children when they first enter nursery and elementary schools and to examine otologically all with a positive personal or family history, whether they show deafness or not. The number of subnormal ears one will find if one looks for them is surprising.

The standards used for adults must be elevated when testing children. Normal children on the average test 10 db better than average normal adults. Our standard of 9 db loss is therefore too low and is defeating in large measure the early prevention of ear conditions. It would be far better to use 6 db, even though this triple the load for the examiner. For practical purposes there may be a 15 db loss at certain frequencies without important implications of trouble. But even in such instances a careful search should be made to account for the variation. A second test should always be made. It will often reveal a slightly better hearing level because of the practice factor.

Up to and even above the age of 6 in many instances, only individual tests are feasible. The residual whispered voice should be heard and words understood by the attentive normal hearing child at a distance of 20 feet in a large quiet room, and at 40 feet in absolute quiet. Have the child repeat simple words it knows; make it a game, and do not prolong the test over a few minutes at a time for fear of tiring the little one. There is a safe margin in favor of this test qualitatively, because if the voice is heard no less than one-third of the average normal distance, it means that the loss of hearing is not over 10 db. This is approximately the standard (9 db) set up for screening the hard of hearing from the normal hearing school children.

What are judged to be the first signs of ear trouble often prove after careful examination to be caused by lesions of long standing. Such evidence makes it imperative to examine every child if we aim to detect early evidences of deafness and to determine a base for future reference. In little babies it is possible to detect deafness by conditioning methods, unintentional or by design. Most babies are automatically conditioned to many sounds and respond by cry and laugh or smile or certain movements on slight sound provocation; that is, if they have no hearing defect. Here accurate audiometric measurements are not necessary. What we desire first, is to know whether or not the baby's hearing is near normal. The greatest obstacle to early detection is a parent's aversion to admitting defect in his or her offspring. A mother nearly always early suspects a real defect, but often delays in acknowledging it. I am dwelling somewhat at length upon this phase of the subject because I am certain it is of great importance from the preventive standpoint.

The standard measure for hearing acuity is minimum audibility. I wish to point out that this is but one criterion, because minimum audibility is not the level at which the human ear habitually senses the ordinary intensities of speech, music, street and other noises. The ear usually ignores faintly audible sounds. A person may hear sounds as well as another at minimum audible intensity, and yet not as well at louder intensities. This symptom may be the earliest sign of certain types of obstructive deafness.

DIAGNOSIS

Detection is of little or no use as a preventive measure without a differential diagnosis and a differential treatment. Differential diagnosis should, of course, play a part in every diagnosis, even if the examiner does not consciously sense the fact that he is making one. There is often an inexcusable laxity in otolgic diagnosis. Note the following commonly used expressions: "progressive deafness," "inherited deafness," "tubal catarrh," "chronic catarrhal otitis media" (OMCC)—whatever that is, "nerve deafness," etc. All of these are indefinite or incomplete diagnostic terms.

It is quite a common practice to make a diagnosis of "otosclerosis" simply because with deafness there is obtained no history or sign of ear inflammation. In my opinion a large proportion of the diagnoses of otosclerosis are false, although the personal and family history, hearing tests, and the deafness, fit into the textbook descriptions of this condition. We obtain in almost one-third of the otosclerosis found at autopsy, no evidence of ankylosis of the stapes although ankylosis of the stapes is the lesion which causes the typical obstructive and progressive deafness of this baffling entity.

Many otologists lose interest immediately after making a diagnosis of otosclerosis. With few exceptions much can be done, particularly in the young, not for cure of the otosclerosis, but for the prevention of the deafness caused by the coincidal lesions which may always precede or accompany it and which are, I believe, at least one factor in the etiology of the condition. The examiner has not finished when he has made a diagnosis of otosclerosis until he has otoscopically, with the unaided eye and with the aid of the magnifying pneumatic speculum, recorded the luster, transparency, congestions, edemas, bulgings, retractions, increased or diminished tensions, increased or diminished movements, scarrings, perforations, adhesions, and calcium deposits, in both the membrana tensa and flaccida, and the movability of drum and umbo, and also abnormal vasomotor phenomena on the drum and promontory. He should make this a routine in every otoscopic examination.

It is generally believed that otosclerosis has no relation to any other disease of the ear. I do not believe this because I find few cases without clinical indications of present or past inflammation in the middle ear. In over fifty cases examined by microscopic serial sections, including slides both of our own and others, positive signs of past inflammatory episodes were found. In no instance were such signs absent.

* "db" is an abbreviation of the word "decibel." A decibel is the standard unit of measure for sound intensity. One decibel is the minimum amount of loudness change that can be detected as such. A decibel is the sensation unit (S.U.) used in measuring hearing capacity.

It has never been proved that consanguinity, which is in all recessive disease an important factor in the increment of familial defects, induces an increase in the family incidence of otosclerosis. The contrary appears to hold in many families. Moreover, although much effort has been expended to prove that otosclerosis is wholly a familial disease and transmitted as such to future generations, as in Mendelian types, there are so many exceptions as to similarity and exact seat of lesion, number in family attacked, number perpetuated, identical age of onset, course, arrest, symptomatology, etc., that it is clear to me that otosclerosis, like some other types of obstructive deafness, is not dependent wholly upon inheritance and that local factors are involved in its etiology and progress.

It is worth noting that routine adult autopsy findings show one in twenty with otosclerotic lesions, not over one-half of whom really suffered from deafness. In children otosclerosis is rare and in early childhood very rare. In the fetus it is still a question if it has ever been identified. Surely a disease wholly dependent upon hereditary influences should show some premonitory sign at an arly age. None has been discovered.

Audiometric measurements are of inestimable value in early differential diagnosis if both air conduction and bone conduction are measured. Air conduction and bone conduction may be within normal limits for the frequencies most used for speech (256 to 3000 inclusive), although one or both are far from normal for some of the lower tones or the higher tones. If this obtains the hearing is not normal, and if only from a preventive standpoint, a differential diagnosis is called for. Audiometric examinations are of value not only as aids to diagnosis but for measuring the time variations in hearing. They are far more accurate for this purpose than tuning forks.

As aids to diagnosis one should always examine the nose, throat and teeth, and often steroscopic x-rays of these structures, blood counts, blood chemistry, tuberculin and Wassermann reactions, and for focal infections.

COMMUNICABLE DISEASES REPORTED

Urban and Rural - January, 1936.

Measles: Total 1029—Winnipeg 638, St. James 60, Flin Flon 45, St. Boniface 36, Garson Village 28, Kildonan East 28, Macdonald 25, St. Vital 25, Archie 16, Transcona 14, Kildonan West 12, Springfield 10, St. Andrews 8, St. Laurent 7, Thompson 7, Kildonan North 6, St. Clements 6, Grey 5, Whitewater 5, Fort Garry 4, Morris Rural 4, Unorganized 4, Wallace 4, De Salaberry 2, Franklin 2, Glenwood 2, Hanover 2, Miniota 2, Montcalm 2, Norfolk North 2, Riverside 2, The Pas 2, Argyle 1, Armstrong 1, Assiniboia 1, Cameron 1, Coldwell 1, Killarney Town 1, Minitonas 1, Portage Rural 1, Rockwood 1, Selkirk 1, Stonewall 1, Strathclair 1, Tuxedo 1, Virden 1.

Mumps: Total 255—Winnipeg 140, St. Boniface 27, Kildonan West 18, Kildonan East 16, Dauphin Town 15, St. James 13, Unorganized 8, St. Vital 3, Carman 2, Harrison 2, Springfield 2, Strathclair 2, Argyle 1, Emerson 1, Fort Garry 1, Killarney Town 1, Ste. Anne 1, St. Clements 1, Tuxedo 1.

Chickenpox: Total 196—Winnipeg 119, Flin Flon 36, Unorganized 12, St. Boniface 5, St. James 4, Brandon 4, Tuxedo 3, Daly 2, Dauphin Town 2, Selkirk 2, Dauphin Rural 1, Elton 1, Kildonan East 1, Neepawa 1, St. Clements 1, Whitemouth 1, Whitewater 1.

Scarlet Fever: Total 169—Winnipeg 85, Flin Flon 9, St. Boniface 7, Louise 6, Argyle 6, Brandon 6, Kildonan West 5, Clanwilliam 4, Manitou 4, Norfolk North 4, Elton 3, Shoal Lake Rural 3, Neepawa 2, Pembina 2, Rosedale 2, St. Clements 2, St. James 2, Thompson 2, Birtle Rural 1, Eriksdale 1, Glenella 1, Kildonan East 1, Lac du Bonnet 1, Pilot Mound Village 1, Roblin Rural 1, Rockwood 1, Saskatchewan 1, Swan River Town 1, St. Andrews 1, Transcona 1, St. Paul W. 1, Unorganized 1, Woodlands 1.

Influenza: Total 82—Whitehead 50, Winnipeg 14, Whitewater 5, Brandon 3, Sifton 3, De Salaberry 3, Louise 2, Shellmouth 1, Carberry 1.

Tuberculosis: Total 58—Winnipeg 6, Unorganized 6, Portage City 4, De Salaberry 2, Fort Garry 2, Norfolk South 2, St. Boniface 2, Albert 1, Bifrost 1, Brandon 1, Charleswood 1, Clanwilliam 1, Cypress South 1, Dauphin Rural 1, Dufferin 1, Grey 1, Harrison 1, Lac du Bonnet 1, Langford 1, Lawrence 1, Lorne 1, McCreary 1, Macdonald 1, Mossey River 1, Pilot Mound Village 1, Pipestone 1, Portage Rural 1, Rhineland 1, Rossburn Rural 1, Selkirk 1, Shellmouth 1, Shell River 1, Siglunes 1, Stonewall 1, Ste. Anne 1, St. Clements 1, St. Laurent 1, St. Paul West 1, St. Vital 1, Transcona 1, Westbourne 1.

Whooping Cough: Total 56—Winnipeg 22, Unorganized 10, Whitewater 8, Morris Rural 6, Louise 2, Springfield 2, St. James 2, Brooklands 1, Transcona 1, Whitehead 1, Roblin Village 1.

German Measles: Total 50—Whitewater 26, Rosser 6, Brandon 5, Cypress South 2, Kildonan West 2, Sifton 2, Whitehead 2, Dauphin Town 1, Kildonan East 1, Kildonan Old 1, St. Boniface 1, Tuxedo 1.

Diphtheria: Total 26—Winnipeg 12, Tuxedo 3, La Broquerie 2, Stanley 2, Unorganized 2, Bifrost 1, Cartier 1, Charleswood 1, Fort Garry 1, St. James 1.

Erysipelas: Total 11—Winnipeg 4, St. Boniface 2, De Salaberry 1, Portage City 1, Rhineland 1, St. Vital 1, Whitemouth 1.

Diphtheria Carriers: Total 6—Winnipeg 6.

Typhoid Fever: Total 2—St. Vital 1, Lac du Bonnet 1.

Amoebic Dysentery: Total 1—St. Vital 1.

Cerebrospinal Meningitis: Total 1—St. Andrews 1.

Lethargic Encephalitis: Total 1—Winnipeg 1.

Trachoma: Total 1—Unorganized 1.

Septic Soar Throat: Total 1—Eriksdale 1.

Venereal Disease: Total 106—Gonorrhea 83, Syphilis 23.

DEATHS FROM ALL CAUSES IN MANITOBA

For the Month of December, 1935.

URBAN—Cancer 36, Tuberculosis 8, Pneumonia 8, Influenza 5, Syphilis 3, Lethargic Encephalitis 2, Chickenpox 1, Erysipelas 1, Typhoid Fever 1, all other causes 170, all others under 1 year 4, Stillbirths 12. Total 251.

RURAL—Cancer 38, Pneumonia 26, Tuberculosis 16, Influenza 7, Diphtheria 3, Scarlet Fever 2, Whooping Cough 2, Lethargic Encephalitis 1, Puerperal Septicaemia 1, Syphilis 1, all others under 1 year 6, all other causes 189, Stillbirths 17. Total 309.

INDIAN—Tuberculosis 8, Measles 3, Pneumonia 2, all other causes 9, all others under 1 year 2, Stillbirths 1. Total 25.

Medical Library
University of Manitoba

A summary of the contents of some of the journals available for practitioners, submitted by the Faculty of Medicine of the University of Manitoba. Compiled by T. E. HOLLAND, B.Sc., M.D. (Man.), F.R.C.S. (Edin.).

"The Practitioner"—January, 1936.

This number contains a symposium on "Winter Ailments" composed of the following articles:

"On the Treatment of Lobar Pneumonia" — by Sir Arthur J. Hall, M.A., M.D., F.R.C.P., Emeritus Professor of Medicine, University of Sheffield.

"Influenza"—by A. J. Scott Pinchin, M.D., F.R.C.P., and H. V. Morlock, M.C., M.D., M.R.C.P.

"The Common Cold; Its Prevention and Cure"—by E. P. Poulton, M.A., M.D., F.R.C.P.; and F. A. Knott, M.D., M.R.C.P., Guy's Hospital.

"The Management of Otitis Media and Sinusitis Complicating the Common Cold"—by Lionel Colledge, M.B., F.R.C.S.

"Acute Sore Throat"—by E. Watson-Williams, M.C., Ch.M., F.R.C.S.E., Bristol.

"Chronic Cough in Children"—by W. G. Wylie, M.D., F.R.C.P., Hospital for Sick Children, Great Ormond Street.

"The Effects of Winter on Chronic Rheumatic Conditions"—by J. Barnes Burt, M.D.

"Winter Dermatoses"—by H. Haldin-Davis, M.A., M.D., F.R.C.P., F.R.C.S.

Further articles in this issue are:

"The Treatment of Stammering"—by Cortlandt Mac Mahon, M.A., Instructor for Speech Defects and Breathing Exercises, St. Bartholomew's Hospital.

"Haemolytic Streptococcal Fever"—by Francis Ind., M.D.

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"The Clinical Journal"—January, 1936.

"Local Anaesthesia in General Practice" — by Norman C. Lake, M.D., M.S., D.Sc., F.R.C.S., Senior Surgeon, Charing Cross Hospital.

"The Treatment of Toxic Goitre" — by J. W. Linnell, M.C., M.D., M.R.C.P.; and R. J. McNeill Love, M.S., F.R.C.S. (Eng.).

"Simple Gastric Disorders and their Treatment" —by Douglas Firth, M.A., M.D., F.R.C.P.

"'Aids' to Hearing: Investigation of the Deaf" —by F. Holt Diggle, F.R.C.S. (Eng.).

"Fractures of the Tibia involving the Knee Joint"—by A. T. Fripp, F.R.C.S.

"Minor Surgery in the Anal Region"—by H. T. Simmons, Ch.M., F.R.C.S.

"The Canadian Medical Association Journal" —January, 1936.

"Avertin"–An Analysis of 1600 Administrations —by Sir Francis E. Shipway, K.C.V.O., M.D., Ch.B., London, England.

"Subtemporal and Suboccipital Myoplastic Craniotomy"—by William Cone, M.D., and Wilder Penfield, M.D., Montreal.

—The operative technique used by the authors is described and well illustrated by drawings.

"Further History of the Care and Feeding of the Dionne Quintuplets" — by Allan Roy Dafoe, O.B.E., M.D., Callander, Ont.

—This interesting article describes the care of the Dionne Quintuplets during their first year of life. Letters of advice and offers of help were received from many countries of Europe and Asia, from Australia and from all over North America. The list of therapeutic measures offered is very amusing.

The report of the birth and immediate care of the infants was published in the "Journal of the American Medical Association" 1934. 103, 673.

"The Treatment of Congenital Syphilis with Stovarsol"—by A. M. Davidson, M.B., Ch.B. (Edin.), M.D. (Man.), M.R.C.P. (Edin.), F.R.C.P. (C), Lecturer in Dermatology, University of Manitoba; and A. R. Birt, M.D., Clinical Assistant in Dermatology, Children's Hospital of Winnipeg.

—Fifty-one cases of Congenital Syphilis treated in the Out-Patient Department of the Winnipeg Children's Hospital are discussed and the advantages of Stovarsol given by mouth are shown.

"Surgical Emergencies in General Practice Exclusive of Trauma"—by Roscoe R. Graham, M.B., Toronto.

"Obesity and its Treatment" — by Walter R. Campbell, M.D., F.R.C.P. (C), F.R.S.C., Toronto.

"Induction of Labour by Rupture of the Membranes"—by E. Murray Blair, Vancouver.

"Is Cardiovascular-Renal Disease Increasing as a Cause of Death in Canada?" — by Madge Thurlow Macklin, A.B., M.D., Medical School, University of Western Ontario, London, Ont.

"An Experimental Production of Coronary Thrombosis and Myocardial Failure" — by G. E. Hall, G. H. Ettinger and F. G. Banting, University of Toronto, Banting Institute.

—This investigation produced coronary thrombosis and myocardial failure in dogs by intravenous injection of acetyl-choline the lesion being comparable to those found in man when coronary circulation has been deficient.

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"The Journal of the American Medical Association"—November 16th, 1935.

"Hysterical Paralysis and its Treatment" — by Abraham Myerson, M.D., Boston.

—A number of case histories is included and treatment described.

"The Choice and Interpretation of Tests of Renal Efficiency"—by R. H. Freyberg, M.D., Ann Arbor, Mich.

"The Journal of the American Medical Association" —January 18th, 1936.

"Bacterial Meningitis" — A Comparative Study of Various Therapeutic Measures—by Carlo J. Tripoli, M.D., New Orleans.

—A study is made of 468 cases and the various therapeutic measures used are discussed.

"Protamine Insulinate" — by H. C. Hagedorn, M.D.; B. Norman Jensen, M.D.; N. B. Krarup, M.D., and I. Worstrup, Copenhagen, Denmark.

—In an attempt to effect absorption of insulin more slowly and over longer periods of time thus emulating the normal secretion of the pancreas, these workers have combined insulin with a protamine, resulting in a levelling of the blood sugar curve and almost complete disappearance of symptoms of insulin reaction, and hyperglycaemia so common with administration of ordinary insulin due to its rapid absorption and quick action.

"Clinical Experience with Protamine Insulinate" —by Howard F. Root, M.D.; Priscilla White, M.D.; Alexander Marble, M.D., and Eliner H. Stotz, B.S., Boston.

—Fifteen patients were treated in Boston, the results of which are given in this paper. The observations of the Danish workers were largely confirmed. Blood sugar levels were found to be evener and more prolonged and hyperglycaemic reactions almost entirely avoided.

The following papers were read before the Section on Urology at the Annual Meeting of the American Medical Association, Atlantic City, June, 1935. An abstract of the discussion which followed is included.

"Role of Anomalies of Kidney and Ureter in Causation of Surgical Conditions"—by Robert Gutierrey, M.D., New York.

"The Embryologic and Clinical Aspect of Double Ureter" — by Allan B. Hawthorne, M.D., Montreal.

"STONE WALLS DO NOT A PRISON MAKE NOR IRON BARS A CAGE."

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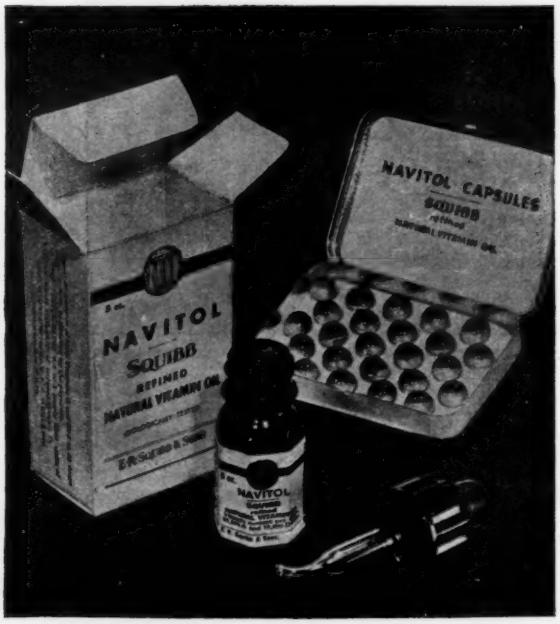

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Clinical Section

*Gastro-Intestinal Symptoms Associated with Diseases of the Urinary Tract

By

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Diseases of the genito-urinary tract give rise to gastro-intestinal symptoms which are of two types, namely, the acute and the chronic. The acute symptom being usually pain, and the chronic nausea, vomiting, anorexia, etc. These symptoms may be so predominant in either case as to overshadow all the complaints, if any, that might be referable to the urinary tract, and unless one is very acutely aware of the possibilities, the real source of the condition is often overlooked.

Cecil reviewed 300 cases who had had complete urological investigation, to determine the frequency of abdominal pain in association with urinary tract lesions, and found that in 28% of 76 cases of stone in the kidney and ureter, that abdominal pain was the presenting symptom; and in 20% some abdominal operation had been performed before the urinary calculi were discovered. While in a series of 26 cases of hydronephrosis 30% had abdominal operations before the source of the trouble was located. The presence of abdominal symptoms also was a confusing factor, but to a lesser degree, in many other types of urinary tract lesions in his series of cases.

Probably the symptom that causes most confusion is pain, and the presence of abdominal pain in disease of the urinary tract, particularly the kidneys, is usually explained as being due to the intimate connection of the sympathetic nerve fibres of the visceral organs and of the kidneys and ureters. To give briefly this connection: The renal plexus of nerves arise by fibres from the solar plexus, the splanchnic nerves, the inferior mesenteric ganglion and probably fibres from the vagus. These nerve fibres run along the renal arteries, anastomose freely, and in their course from several small ganglionic masses. The nerves enter the hilum of the kidney, branch with the blood vessels, and end in the glomerular capsules, tubules and between the epithelial cells. So through the superior and inferior mesenteric ganglia, the renal plexus is connected with the sympathetic nerve supply of the stomach and intestines. Anastomoses also have been shown to exist between the renal plexus and the aorticomesenteric ganglia, which supply the stomach. Animal experimentation has shown that stimulation of the sympathetic nerve fibres to the kidney and upper ureter, cause disturbances of the intestinal masculature.

The distressing symptoms of abdominal pain, associated with persistent nausea and vomiting, which occasionally occur following catheterization of the ureters, pyelography or acute obstruction from stone, are ascribed by Colby to a reflex along the intimate sympathetic connections of the kidneys, ureters and gastro-intestinal tract.

The urinary tract may give rise to gastro-intestinal symptoms in two other ways; first, they may be obstructive in character and result from the pressure of large renal tumors on adjacent structures, such as large hydronephroses, pyonephroses or neoplasms. I will also include under this heading the rarer cases where gastric obstruction or jaundice may be present, due to traction on the second portion of the duodenum and adjacent structures. Such a case was reported by Scholl. Here the patient presented with jaundice and vomiting; on the establishment of renal drainage, by means of ureteral catheterization, the jaundice and other symptoms were relieved—withdrawal of the catheter again produced the symptoms. Nephrectomy, later, gave a permanent cure of the gastro-intestinal symptoms.

Secondly, gastro-intestinal symptoms may be manifestations of a failing renal function, that is, anorexia, nausea, vomiting and epigastric pain. This failing renal function may be due to several causes, the most common being, Bright's disease and prostatic obstruction; or more rarely, bilateral pyelonephritis, bi-lateral renal tuberculosis, polycystic kidneys, etc., or any lower urinary tract obstruction. Braasch says, "when a male patient aged more than 40 years comes to the physician with headaches, loss of appetite, occasional nausea or even vomiting, with pain referred to the epigastrium, it behooves him to make a rectal examination and to determine whether there is any evidence of prostatic enlargement or other evidence of urinary retention.

I would like to present a few cases illustrating to some extent the types of gastro-intestinal symptoms produced, and how unreliable or misleading these symptoms may be when an effort is being made to ascertain the exact location of the lesion.

1.–F. H., male, aged 47.—On July 18, 1930, complained of acute pain in his lower abdomen, with nausea and vomiting. He also thought his bladder was full and catheterization showed about two ounces of urine, which contained pus 1. I had previously seen this patient and knew he had a pyuria of a few cells from a chronic prostatitis. Examination showed a temperature of 100, pulse 98. There was also very definite tenderness in the right lower quadrant, over McBurney's point, with considerable resistance f the abdominal wall.

* Read at the Post Graduate Course on Gastro-Enterology, Manitoba Medical College, February, 1936.

He was hospitalized and his white blood count was 21,000. The internist and a surgeon were called in consultation and advised operation. At operation a comparatively normal appendix was removed. The following 24 hours the patient did not void, and catheterization showed no urine to be present in his bladder. An x-ray of the kidney, ureter and bladder showed a shadow, 1x½ cm. in diameter, in the lower right ureteral area.

Cystoscopic examination showed an obstruction about 3 cm. from the right ureteral orifice, which was passed with some difficulty, and there was about 30 cc. of urine in the kidney pelvis. The catheter was left in for 48 hours. The day following its removal the ureteral calculus was passed. Cystoscopy later showed this patient to have only one kidney.

COMMENT

This case illustrates the ease that one encounters in making a wrong diagnosis, even with symptoms that should have led us on the road to a correct solution; that is, the stranguary with very little urine in his bladder.

There are probably very few surgeons who have not sometime during their career removed a normal appendix, in the presence of lesions of the right kidney, usually calculus disease. Given a case where the diagnosis of acute appendicitis is in doubt, I feel that the majority of them turn out to be calculus disease of the right kidney or ureter; but in spite of that, it is far safer to remove some normal appendices than to leave one to rupture.

This case also is of interest in that it presents one of the unusual anomalies of the urinary tract. that is, a congenital solitary kidney. This occurs, according to Ballowitz who found it present in 12 cases in a collected series of 28,423 autopsies, or 1 in about 2400 cases.

2—M. M., male aged 13. Was first admitted to the General hospital on September 16, 1931, complaining of colicky pain in the left lower quadrant associated with nausea and vomiting, he also complained of constipation. He was discharged with no definite diagnosis. On November 20, 1931, he was re-admitted with similar complaints, and on December 1, his appendix was removed and there were some adhesions separated in the left side of his abdomen. He was discharged on December 18, 1931.

On December 26, 1931, he was re-admitted with a history of going to bed the previous night with slight headache and a pain in the lower left abdomen, he wakened during the night with the pain more severe, nausea and later vomiting. He was discharged on January 7, 1932, with a diagnosis of Gastro-enteritis. On February 2, 1932, he was re-admitted with identical symptoms, and a diagnosis of Dibothrocephalus Latus was made. The worm was obtained intact. He was discharged February 6, 1932.

On February 16, 1932, he was re-admitted with similar complaints. The history taken gives a good description of his pain: "The pain is sharp, severe and colicky; it originates fairly repidly and subsides gradually; commencing in the lower left abdomen, it does not radiate." He was discharged again on February 18, 1932, with a diagnosis of constipation and impacted faeces. During all his admission his urinlaysis was normal.

On May 30, 1932, he was re-admitted with the same symptoms, but with a note from the admitting doctor that a renal lesion was suspected. An intravenous urogram was done which showed a hydronephrosis on the left side. On June 8, I operated on this boy, finding a small vessel associated with a dense fibrous band, crossing and exerting pressure on the uretero-pelvic juncture. This was ligated and cut. The pelvis immediately decreased somewhat in size. A nephopexy was done, and slide II shows the kidney pelvis, three months later. This boy has been completely well since then.

COMMENT

A more illustrative case could hardly be found; where the abdominal pain caused by a renal lesion was thought to be due to many different abdominal conditions, and one abdominal operation performed before anyone thought of investigating the urinary tract. It shows clearly the importance of never neglecting the urinary tract in all cases of abscure abdominal pain, even if the urinary findings are normal. This case is rather unusual, in that the right kidney is more often at fault in causing abdominal symptoms than the left, in that the sympathetic connection has been demonstrated to be closer.

3—Male aged 44. Was admitted to the hospital on April 15, 1929, complaining of haematuria, loss of weight, general ill health and dyspepsia. In August, 1928, he began having upper abdominal distress after eating and feeling generally below par. This continued, and in January, 1929, he consulted an internist, who did a thorough physical examination and had a Barium series done. At that time he had lost approximately 15 pounds in weight. No cause for his condition was discovered. On April 12 he passed practically pure blood from his urethra, this blood continued and associated with it was a frequent desire to urinate. About the second day of the haematuria he experienced difficulty in voiding, plus suprapubic pain, and at that time had a dull aching pain in his right loin. He had lost approximately 25 pounds in the last year.

On examination the patient had apparently lost considerable weight, was anaemic in appearance and appeared sick. The abdomen was slightly tender in the right upper quadrant and suprapubic area. Palpation was difficult due to resistance, but an indefinite mass was palpable in the right upper quadrant. There was no suprapubic dulness. The blood pressure was 130 systolic and 95 diastolic. Urinalysis showed the urine to be grossly bloody; specific gravity was

1010; acid in reaction; there was albumen present but no sugar was found. Microscopically an occasional granular cast was seen, and there were many red blood cells. The blood urea nitrogen was 26.6 mgs. per 100 cc. of blood. The blood examination showed 4,100,000 red blood cells and 10,800 white blood cells. The haemoglobin was 70%. Roentgenograms of the kidneys, ureters and bladder were negative.

The cystoscopic examination showed the bladder to be practically filled with a well organized blood clot. This was broken up as much as possible and a considerable amount removed. Three days later, under sacral anaesthesia, the remainder of the blood clot was practically removed. The urine excreted from both ureteral orifices was clear. Indigo-carmine appeared in 4 minutes from the left side in good concentration; it appeared from the right side in 8 minutes, with about one-half of the concentration. A pyelogram done on the right side showed a marked distortion of the pelvis and calyces. Roentgenograms of the lungs, done as a routine pre-operative procedure, showed multiple metastatic growths. The patient was allowed to go home, and from there he entered a "cancer cure" institution where he stayed for over a month, but in spite of that he gradually became worse, and died in October, 1929.

COMMENT

This shows a case where the gastro-intestinal and general symptoms were the predominant feature until the haematuria appeared. Repeated questioning failed to elicit any previous history of haematuria. This patient's general examination revealed no cause for the symptoms; but coupled with his weight loss malignancy should have been suspected. In all cases where malignancy is suspected, and the source not readily discovered, the urinary tract should always be investigated — particularly the prostate and kidney.

In renal neoplasms we have what is called a triad of symptoms, namely: tumor, pain and haematuria—any one or all of these symptoms may be present, but one should never forget that in the presence of haematuria and a mass on one side, never neglect to palpate the opposite renal area, as polycystic kidneys may produce similar symptoms. The haematuria of renal tumor is total in character, usually coming on suddenly, lasting a short while only, stopping suddenly, and recurring at frequent intervals.

I would also like to emphasize the importance of pre-operative routine roentgenograms of the chest to exclude metastases. This case was apparently operable as regards the urinary tract findings.

4—W. W., male aged 23. First seen on October 4, 1930, complaining of frequency for the past eight months and some terminal haematuria for two months. At that time the general examination was negative. The urinalysis showed a considerable amount of pus, and on staining, many acid-fast bacilli were found. He and his mother were told of his condition and hospitalization for investigation was advised. On November 11, 1935, I was called and asked regarding this boy. He had been in bed for a week complaining of headache, nausea and vomiting, for which he was being treated. On being seen by another doctor his mother gave the history, that he had been told five years before by me, that he had tuberculosis of the urinary tract. On admission to the hospital his blood urea nitrogen was 215 mgs. per 100 cc. of blood. The urine showed a specific gravity of 1015, acid in reaction, albumen present, no sugar; microscopically there was pus IV and many acid-fast bacilli. His nausea, vomiting and headaches continued. On November 23, 1935, his blood urea nitrogen was 232 mgs. and his creatinine 13.3 mgs. per 100 cc. of blood. He died on November 27, 1935, in convulsions.

COMMENT

This case illustrates several points, one of which has no great scientific interest; but this boy, after being told that he had tuberculosis of the urinary tract, entered as a medical student and during his course, repeatedly stained his urine, always finding the acid-fast bacilli; but still carrying on.

Here we are dealing with the gastro-intestinal symptoms, due to a failing renal function, which were sufficient to overshadow the real diagnosis, and he was treated palliatively for a week before the real cause was discovered. This boy probably in 1930, had a unilateral renal tuberculosis, as only about 10% of the cases showing open renal tuberculosis are bi-lateral; the remaining 90% of unilateral cases are curable in about 70% of cases, by surgical intervention.

5—J. G. M., male, aged 70 years. Was referred to me on March 6, 1931, complaining of headache, anorexia, nausea, vomiting and inability to void. He gave a history of being treated for pyelonephritis and of bed wetting for the past 3 years. He had lost about 30 pounds in the past three months. The examination showed an obviously very ill and anaemic male. The deep reflexes of the legs were absent and the pupils frozen. There was almost complete retention. There was a to and fro murmur over the aortic area. The prostate was normal per rectum. The urinalysis showed a specific gravity of 1010, albumen present, no sugar; microscopically it was loaded with pus. He was hospitalized. The blood urea nitrogen was 122 mgs, and the creatinine was 6.0 mgs. per 100 cc. of blood.

COMMENT

In this case, the diagnosis of which was of course obvious and not confusing, we have another illustration of gastro-intestinal symptoms due to a failing renal function; brought on by a lesion in the posterior roots of the spinal column, with resulting palaysis of the bladder. These so-called cord bladders are often

neglected, and are at all times a difficult problem in the management of late lues of the central nervous system.

These same symptoms are not unusual in men with prostatic obstruction, although luckily not a rule so severe or advanced; but it is not unusual to see patients who complain of anorexia, constipation and dryness of the mouth, the cause of which is an early so-called chronic uraemia.

*Chronic Duodenal Stasis

By

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This is a clinical entity, not uncommon but generally overlooked; it is also described as chronic duodenal ileus or chronic duodenal obstruction; compression of the duodeno-jejunal junction between the tight root of the messentery and the lumbar spine is the most familiar cause of the duodenal statis, which, as a permanent or intermittent state, constantly accompanies the condition, whatever its origin may be.

Etiology: Most of the recorded cases, coming as they do from surgical clinics, present duodenal obstruction at the duodeno-jejunal junction; here enteroptosis is usually found, often associated with lack of supporting fat or defective bodily posture—coils of small bowel or a mobile caecum and ascending colon slip into the pelvis and drag on the mesenteric attachment so that, especially if the mesentery be short, the bowel is compressed against the spine and statis of its contents results. In other cases, Lane's Kink or chronic inflammatory thickening of the root of the mesentery is present; in still other cases, tuberculosis or even carcinomatous glands in the same position may similarly narrow the gut.

But in recent years, many other types of duodenal statis have been noted, only a few of which can be touched on here. Feldman, particularly, has described the "redundant duodenum," where the first part instead of bending down to the right and posteriorly from the cap to continue as the second portion, elongates more or less horizontally from the cap for some centimetres and is supported by the hepato-duodenal ligament before dropping abruptly into the descending portion of the duodenum. This lengthening of the superior portion may cause an anomalous looping of the gut and so give rise to duodenal stasis with sometimes associated ulceration. Again, stasis may occur about the junction of the second and third portions of the duodenum; Dr. McMillan, of Winnipeg, thinks this latter may be due to undue laxity of the duodenum in the erect position, so that the cap and descending portion sag down unduly, with resulting kinking. Congenital bands and adhesions, sometimes from the gall bladder, sometimes from the pancreas, sometimes connected with the colica media artery crossing the second part of the duodenum, are evidently responsible in other cases.

Weinbren has recently described a number of cases of so-called right-sided duodenum inversum, in which the third part of the duodenum, instead of turning to the left and upwards, curves round to the right and so kinks, as it comes to lie as high as or higher than the duodenal cap before passing into the jejunum.

There is, lastly, no doubt that one form of duodenal stasis exists which is not obstructive; a dilated duodenum, generally in the second part, has been found repeatedly at operation by Wilkie, Judd and others, with no kinking or stenosis present; this variety evidently results from some form or neuro-muscular derangement and is analogous to the megacolon found in Hirschsprung's disease.

Frequency: There is, of course, room for wide difference of opinion in regard to the frequency of duodenal stasis; it can hardly be a rare condition, as I have personally seen in private practice 13 cases in the last year, two confirmed at operation and the others reasonably certain from the combined clinical and radiological examinations. Of these 13, 11 occurred in women; the average age was 32, varying from 21 to 48 years; the average weight of 7 of the number was 104 lbs., indicating sufficiently the type of individual usually the victim; the average duration of symptoms was 3½ years, ranging from 3 months to 10 years in individual cases. In 4 the appendix, and in 1 the gall bladder had been removed without benefit. All the patients were carrying on their duties and had not been confined to bed, except occasionally for a few days at a time, though in several cases additional help has been necessary in the house and one man had done little for many months.

Symptoms: There seems to be little difference in the sypmtomatology of the different types of duodenal stasis above described. Epigastric fullness and bloating, sometimes associated with marked nausea, comes on soon, generally within half an hour, after meals, though in three of my cases the distress did not appear till 2 to 3 hours after food, some relief being experienced by eating again. The fullness and discomfort last a varying time, sometimes for an hour or two, are more marked after a large or indigestible meal, are helped by belching of gas and especially by lying down. Two of my patients had discovered that they got relief by kneeling in the knee-chest position and pressing with the hands below the navel in an up and back direction. The epigastric distress sometimes amounts to a definite pain which is occasionally severe, sharp or cramplike; it may come in spells of a few days' duration, precipitated by overwork, worry or indigestible large meals, but later it tends to recur practically every day.

*Read at the Summer School of the Vancouver Medical Association, June, 1935.

Regurgitation of mouthfuls of food or of sour liquid is frequent after the fullness has lasted for some time; occasionally, severe and recurring vomiting of considerable quantities of liquid, sometimes persistently bilestained, may occur at intervals of weeks, and these so-called "bilious spells" may persist for many hours or even for a day or two.

Constipation, sometimes severe, was specially noted in half my cases, though no marked relief from the epigastric distress was obtained when the bowels moved; discomfort, however, along the colon complained of by three was relieved by action of the bowels. Lack of appetite was generally present with inability to take a fair-sized meal; loss of weight was almost constantly met with, amounting to 20 lbs. on an average. Spells of diarrhœa, noted by several observers, were not met with in any of my cases.

Headache was complained of by seven; in two there was a family history of migraine; in six, the headaches antedated the digestive symptoms by many years, were mainly of the migrainous type, ending in vomiting, had become more frequent with the onset of indigestion; in one patient, daily headache had been present for 14 months. The headaches were apt to come when the patients were overtired, either from worry or overwork, but also after indigestible food and especially after sweets and chocolates. I found no evidence that animal food specially precipitated headaches, as has been claimed by some writers; in women, the headaches might be associated with menstruation but were usually independent.

All the patients complained of being tired; most were tired all the time, two only at the end of the day's work. Many were nervous, irritable and rather depressed, so that superficially they might pass readily as neurasthenics.

On *physical examination*, the patients were usually rather pale and obviously undernourished, at times quite emaciated, and the blood pressure was low. Three were noted as of normal build; all the others were obviously enteroptotic with the usual poor muscular development, long narrow chest and sagging belly. Definite fullness in the lower epigastrium and just below the navel was generally present with some tenderness; often splashing could be elicited well below the navel some hours after food. Hayes claims, by steady pressure upwards and backwards on the abdomen below the navel for some minutes, to be able to empty the duodenum into the bowel below and so to remove the distension previously obvious. It is practically impossible to make out on physical examination the dilated duodenum, though more marked sensitiveness and distension in the midline or immediately to the right of the navel may arouse one's suspicion of the condition. A test breakfast gave in every case free hydrochloric acid, within normal limits.

It should be specially emphasized that in the history, periods of comparative or absolute well-being alternated with spells of digestive distress and lassitude, justifying Friedenwald's description of chronic *intermittent* duodenal stasis with, presumably, periods in which even the x-ray would show no abnormality.

Diagnosis: A definite diagnosis can be made only by x-ray examination, but a provisional diagnosis of duodenal stasis was made in several of my cases before confirmation was sought. The combination of recurring headaches, apparently migrainous in type, with rather indefinite epigastric distress, is particularly suggestive; the history of relief obtained by kneeling in the knee-chest position or possibly by pressure below the navel may also help, especially when the age, sex and build of the patient is considered. Fullness and bloating after meals naturally suggest cholecystitis, but the slight build and youth of the average patient are against this diagnosis. In the exceptional case, duodenal ulcer is suggested by distress or pain, coming an hour or so after meals, with partial or complete relief by food; there is rarely a typical clear-cut history of attacks with complete relief in the interval, and in the attack, soda may fail to give relief, inducing, some suggest, very readily alkalosis. The possible combination of duodenal ulcer with duodenal stasis must be remembered.

Every case of obstinate migraine, especially with somewhat anomalous digestive symptoms, must be reviewed from the duodenal statis standpoint. It would seem that the old idea of intestinal intoxication, with absorption of hypothetical toxins from the colon, now largely discredited, has its justification in the frequent association of weariness, depression and headache with duodenal statis. It is well known that high intestinal obstruction with persistent vomiting will give rise to severe toxic symptoms, dehydration and altered chemistry of the blood, while Brown, Eusterman and others have reported toxic nephritis in duodenal obstruction.

X-ray Examination: In suspected cases of duodenal stasis, and indeed as a routine in order to pick out unsuspected cases, the duodenum must be examined flouroscopically both in the erect and recumbent positions, the oblique and lateral views being often particularly helpful. Too much attention has been concentrated on the duodenal bulb because of the frequency of duodenal ulcer, and too little has been paid to the rest of the duodenum. In mild cases, duodenal stasis may be obvious only in the erect position and can be readily missed, especially if fluoroscopic examination, which shows clearly antiperistaltic movements, be omitted. While variations occur in the x-ray pictures according to the different etiological factors present, it may be said in general that diatation and stasis will be demonstrable in the duodenum, with frequent peristalic and antiperistalic waves, carrying the duodenal contents forwards and backwards from pylorus to the site of interference. (Occasional antiperistalic waves may be seen over the duodenum in the normal individual, it is said.) Stasis should generally be

shown in plates taken 1½ to 2½ hours after the barium meal; usually in 5 hours, the stomach and duodenum are empty (in two of my cases there was considerable residue at 5 hours) though in marked cases requiring surgical interference, a considerable residue may remain. An irritable duodenal cap or one showing definite radiological evidence of ulcer may accompany the duodenal stasis. Visualization of the gall bladder by Graham's method will help in the differential diagnosis.

Treatment: Two of my 13 cases were operated on. One, a strongly built man of 21, had enjoyed good health till a year previously, when after severe right-sided abdominal pain with vomiting, for 12 hours, an appendix said to be "only moderately affected" was removed. Thereafter, he was nauseated after each large meal, had lost 40 lbs in weight; headaches present in boyhood had recurred nearly every day and five quite severe attacks of abdominal pain localized to the right iliac fossa had occurred, associated with gas and vomiting. An x-ray taken a few months previously missed the duodenal stasis; a second radiological examination, with duodenal stasis specially considered, showed definite stasis of the second portion of the duodenum—a finding confirmed at operation, when an inch long stump of the imperfectly removed appendix was found, with also marked dilatation of the descending portion of the duodenum, due to a tight band (? associated with the colica media artery) crossing the duodenum in this position. The patient has been very well since the operation, which involved removal of the appendix stump and a duodeno-jejunostomy.

In the other case, a duodenal ulcer with a diverticulum of the first part complicated the marked dilatation of the second portion of the duodenum; here, too, Dr. Thorlakson performed a duodeno-jejunostomy. A third patient, an emaciated, restless little woman who refused to follow medical treatment, was advised operation but went instead to visit her mother in Vancouver, where a physician was able to put her to bed, with considerable improvement in her condition. Unfortunately, I do not know if this improvement has been maintained since her return to Winnipeg.

Wilkie states that a drainage operation in cases of duodenal stasis due to a neuro-muscular derangement, without any mechanical impediment to the flow, is relatively ineffective.

The other 11 cases were treated on medical lines which naturally had to be adapted to the individual circumstances. No doubt, in most cases, six weeks' rest in bed with the foot raised 10 to 12 inches, would have been advisable, combined with a smooth feeding-up diet and the prone position, or knee-chest, after meals. But actually, unless the patients feel quite disabled, this ideal treatment is seldom practicable. Early hours, more rest in general, help in the house in some cases, lying down in the prone or knee-chest position after meals, the tilting of the foot of the bed some 10 or 12 inches, five smaller meals of the smooth feeding-up variety, liquid paraffin for the bowels, adalin or medinal for sleeplessness, brief abdominal exercises of the simple non-tiring type, possibly better adjustment to personal worries and problems—these measures are useful in the milder cases.

In an attack of abdominal distress or pain the knee-chest position with pressure on the abdomen up and backwards below the navel may help, and in severe attacks the stomach or duodenal tube may be used.

I give briefly the outline of one case with its medical treatment:

Mrs. C. G. M., age 24, seen in March, 1932. Father suffered from sick headaches till about 50. She, herself, had suffered from sick headaches since childhood, which had got much worse in the previous three months, coming every ten days—first nausea, on one side or other, the head, mostly over the temple, begins to ache, with blurring of vision—sometimes the headache is so bad she could bang her head. She usually vomits after three to four hours—solid food, if present, then bile; there is no pain in the spell, which develops especially with chocolates, greasy and fried things, being quite independent of menstruation. She complained also of general swelling of the abdomen, usually within half an hour to an hour after food, lasting for half an hour or longer; there were sometimes gas pains relieved by passage of gas, mostly downwards, and there was slight constipation.

The general physical examination then was quite negative. She was healthy looking and youthful in manner and appearance, but test meal was normal and a gastro-intestinal x-ray was given as quite negative.

She reappeared in June, 1934, said that she was quite well while carrying her only child, which was born in June, 1933, but after that the bilious spells recurred about once in two weeks and were very severe. She still has to avoid cabbage, raw vegetables and fried stuff to help to prevent the bloating after meals, and she finds, too, much sweet stuff also will bring this on.

At this time it was noted that she had a rather long narrow chest with some sagging. The gall bladder visualized normally and the duodenum was specially examined. The second portion was found to be somewhat dilated and there was definite "slushing" movement in the second portion, with stasis at the junction of the second and third portions.

She had been working hard, without help, and she secured help in the house, went to bed early, rested an hour and a half in the afternoon; the foot of the bed was raised ten inches, and she was put on a smooth feeding-up diet, five smaller meals a day with no chocolates or sweets.

She was seen again at the end of March, 1935, when she had gained seven pounds in weight, was feeling much better in spite of a hard winter with the baby sick and her mother ill. She kept

the maid for about four months and adhered closely to the diet till Christmas, since when she has relaxed a little. She now gets the bilious spells only once a month, independent of menstruation, and has no indigestion. It should be noted that after meals she usually lay on her stomach and that the gastric symptoms soon after meals have entirely disappeared.

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